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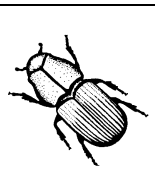
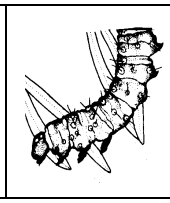

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White Pine Blister Rust Canker Expansion on Improved Western White Pine in Northern Idaho: Implications for the Management of Rust Resistant Stock

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SUMMARY

Nearly 200 white pine blister rust cankers on 48 rust resistant (F2) western white pines in a severely infected northern Idaho plantation were monitored annually for 10 years. Canker expansion ("growth") rate toward the bole was approximately 2.0 inches per year. Cankers closer than six inches to the bole had greater than 90% probability of reaching the bole while cankers greater than 24 inches from the bole never reached the bole. Sporulation was intermittent, but cankers continued to actively expand regardless of sporulation. Cankers near the bole on branches that were dead beyond the canker also continued to grow toward the bole. Infection levels in young stands can change dramatically within a few years, and mortality in severely infected stands can be very high. Trees can have healthy looking crowns and continue to grow several years in spite of being almost completely girdled.

INTRODUCTION

Populations of western white pine (*Pinus monticola* Dougl. Ex D. Don*)* have been dramatically reduced since the introduction of *Cronartium ribicola* J.C. Fisch. in Rabh., the pathogen that causes white pine blister rust, a century ago. Nowhere has it caused more damage than in northern Idaho, where western white pine was a dominant species on about five million acres of highly productive, mid-elevation forest lands. Fortunately, a small percentage of the white pine population was found to be resistant to the rust

disease, and an aggressive tree improvement program was initiated to select and concentrate those resistance genes (Mahalovich 2010).

Genetic resistance forms the basis for white pine reforestation with the goal of restoring the species on sites where it has been severely depleted by white pine blister rust. Planting was started in the mid-1970s, and about 170,000 acres have been planted to date on federal lands in Idaho, Montana, Oregon and Washington (Mahalovich 2010). Most planting stock now is from second generation crosses (F2) of parents exhibiting phenotypic resistance[1].

In western white pine plantations monitored over 30 years, F2 plantations have performed well compared to those of non-resistant stock (Fins et al. 2001) and many show little infection. However, infection of F2 stock has been found to be highly variable, and in some stands as many as 80–90% of the trees may be infected (Fins et al. 2001, Muller 2002, Kearns et al. 2012).

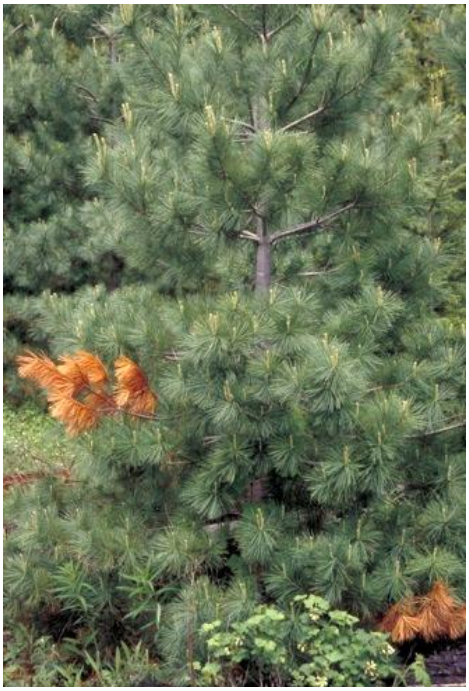


Figure 1. Western white pine with characteristic "flags" caused by white pine blister rust cankers girdling branches.

Infection of white pines can only occur by spores produced on an alternate host, usually gooseberries or currants (*Ribes* spp.) (Miller et al. 1959). Under moist conditions, these spores land on the needles, germinate, and enter the host through the stomata. The fungus then grows within the vascular tissues into the bark, entering branches and stems. The fungus grows down the branch toward the main stem, creating an expanding canker[2] that often girdles the branch. This kills the branch beyond the canker and creates a distinctive red "flag" of dead needles (Fig. 1). After the fungus reaches the stem, it continues to spread and can girdle the stem and cause top-kill or complete tree mortality.

A significant improvement in the survival of unimproved western white pines was achieved when trees were pruned of their lower branches before cankers could reach the bole (Hungerford et al. 1982, Hagle and Grasham 1988, Schwandt et al. 1994). Therefore, pruning techniques were developed and routinely implemented in young plantations of non-resistant trees (Fig. 2) (Hagle et al. 1989, Schnepf and Schwandt 2006). Pruning was also applied in F2 plantations after surveys of young F2 plantations in 2000-2001 (Muller 2002) revealed that many had higher infection levels than expected (Hoff et al. 1973, Bingham et al. 1973).

Figure 2. Pruned western white pine stand.

Pruning guidelines have been based on results from non-resistant trees on which infections originating within a few inches of the bole are very likely to reach, enter, and girdle the bole, eventually killing the tree. On the other hand, most infections farther than 24 inches from the bole rarely reach the bole (Harvey 1967, Slipp 1953, Lachmund 1934). Operational pruning guidelines call for pruning trees if the nearest canker is 6–24 inches from the bole (Hagle et al. 1989, Schnepf and Schwandt 2006, Schwandt and Kearns In press). The assumption has been that infections within six

[1] Due to its early success, the F2 stock has become known as "rust-resistant white pine."

[2] "Canker" in this paper follows the common use of the term to refer to the area of discolored, swollen, and dead tissue associated with a blister rust infection.

inches will likely grow into the bole before a stand can be pruned, and those beyond 24 inches will be non-lethal regardless of treatment.

Two factors are crucial for predicting tree death from white pine blister rust: 1) the probability of bole infection from branch cankers and 2) the rate of canker expansion toward the bole. Neither of these is known for improved F2 stock. Therefore, the main objective of this study was to determine the probability of bole infection from branch cankers at different distances from the bole in F2 stock. Specifically, the study was initiated to determine if the relationship in F2 white pine was different enough from that of non-resistant stock to warrant a change in management guidelines. A second objective was to determine the rate of branch canker "growth" or expansion toward the bole for F2 stock. Earlier work in non-resistant western white pine showed that the mean canker expansion rate was about two inches per year and was positively correlated with branch diameter (Buchanan 1938, Harvey 1967, Slipp 1951).

This study was not designed to compare infection and mortality rates for improved western white pine populations, or stands. This information is provided by other studies (Fins et al. 2001, Muller 2002, Kearns et al. 2012). But other aspects of the damage caused by cankers, such as bole girdling rates and mortality rates of infected trees, were evaluated to the extent possible.

METHODS

The stand (id A7001000005) chosen for study is located on the Coeur d'Alene River Ranger District, Idaho Panhandle National Forests, near Fourth of July Pass. The 30 acre site was planted in 1984 with a mix of western white pine, western larch (*Larix occidentalis* Nutt.) and western redcedar (*Thuja plicata* Donn ex D. Don). One hundred eighty-six F2 white pine seedlings from seed from the Moscow (R.T. Bingham) seed orchard were planted per acre. Surveys for white pine blister rust incidence were conducted in 1995 and 1997.

Field Procedures

Forty-eight F2 white pines distributed throughout the stand were selected for annual monitoring of rust cankers. Twenty-seven trees were tagged and measured in 2000 and the rest in 2001. On each tree, 2–5 cankers were selected and tagged to monitor canker expansion. Cankers were indiscriminately selected from all cankers on a tree within six feet of the ground, and usually included cankers on secondary and tertiary branches as well as primary branches.

A total of 191 cankers were individually tagged and lightly scrubbed with water to aid in the identification of the discolored canker margins (Fig. 3.). Two cankers were added during the study to replace cankers

Figure 3. Branch canker before (left) and after (right) light scrubbing with water showing the yellow/orange leading edge of a canker about 5 inches from the bole.

lost due to broken branches. Thumb tacks were placed at the leading edge of the canker margin, as indicated by discoloration and swelling of the bark. Initial canker data included: distance from the leading edge of the canker to the bole (distance out); type of branch—primary, secondary or tertiary; branch height; internode year of branch canker; presence of sporulation (aecia)(Fig. 4.); branch diameter at the bole; branch condition (live or dead); and for all bole cankers, percent of the bole girdled. The following tree data were also collected: tree diameter at breast height (DBH), height, age, and location.

Figure 4. White pine blister rust canker with characteristic sporulation (aecia).

All trees were re-examined annually in April or May from 2001–2010. Thumb tacks were placed on the new leading edge of the cankers (Fig. 5), and the following data were collected: distance of canker expansion from the prior year tack; presence or absence of aecia; branch diameter at the bole; and branch condition (live/dead). A partial exam was conducted in 2012 to record tree mortality along with percent of bole girdled for trees that had not yet died.

Statistical Analyses

The SAS statistical program (SAS version 9.1, SAS Institute Inc., Cary, North Carolina) was used to analyze probability of bole infection and canker expansion rate. The GLIMMIX procedure was used to calculate probability of bole infection for branch cankers starting various distances out from the bole. Several model runs were made to determine what variables were associated with bole infection. This paper reports on the model run made with only year and distance out as variables, to provide a curve of the probability of bole or branch collar infection based on easily measured criteria. Canker expansion rate was also analyzed using the GLIMMIX procedure. Simple summaries were made to report girdling rates and other results from the study.

Figure 5. Small western white pine branch with a series of tacks showing annual branch canker expansion toward the bole.

RESULTS AND DISCUSSION

Although some rust infections may occur nearly every year, it appears that many infections occur during a single “wave year” when environmental conditions are especially favorable. Lachmund (1933) documented this phenomenon in western white pine and found most white pine blister rust infections occur on one-year old foliage, and lesser amounts occur on current year foliage or

foliage two or more years of age. We found 50% of our monitored cankers on the 1995 internode, 28% on the 1994, 8% on the 1993, and 14% on the 1996 internode. These numbers closely approximate what would be expected from Lachmund's findings if a "wave year" of infection had occurred in 1996.

The 1995 incidence survey found less than 30% of the trees had rust infections, but the survey two years later found infection had increased to 72%. In the 1997 survey, many incipient "spot cankers" were observed. Spot cankers are small, swollen, orange-colored spots typical of very young infections, usually at the base of a needle fascicle (Fig. 6). By the time plots were installed in 2000 and 2001, some trees had dozens of cankers, and infection was estimated to be above 90 percent.

Figure 6. Yellow/orange "spot cankers" develop 1–3 years after needle infection.

Table 1 shows the location of the most lethal canker (bole vs. branch) and the effects of disease on tagged trees over the 12 years of the study. Twenty trees had bole cankers when the study was fully in place in 2001. The remainder had branch cankers only. By 2007, 92 percent (44 of 48 trees) had bole infections, and two of these trees had died. When the study was terminated in 2012, the boles of all trees had become infected and 21 trees had died from stem girdling by blister rust.

Table 1. Location of most lethal canker and mortality of sample trees over 12 years of monitoring.

Year	Live Trees		Dead Trees	Total
	Branch	Bole		
2000	18	9	0	27
2001	28	20	0	48
2002	23	25	0	48
2003	19	29	0	48
2004	12	36	0	48
2005	7	41	0	48
2006	6	42	0	48
2007	4	42	2	48
2008	1	38	9	48
2009	0	35	13	48
2010	0	33	15	48
2012	0	27	21	48

These results show that F2 trees can be severely damaged if they are infected when young (±14 years old in this case). While results may not be representative of all F2 populations, they do show how the disease progresses in young infected F2 trees. Twenty-one of 48 infected trees (44%) were killed by the rust during the 12 years of monitoring. The remaining trees all had bole infections, and most were over 80% girdled. Given the very high severity of infection in the tagged trees (i.e. dozens of cankers per tree), the high level of mortality in F2 stock might be expected. There is some evidence that canker tolerance

(expressed as persistent survival and tree growth in spite of active cankers) may be an inheritable resistance mechanism (Mahalovich 2010), but in stands like this with very high infection levels it may be over-ridden.

Probability of Bole Infection

The first objective of the study was to determine the probability of bole infection from branch infections that occurred at different distances from the bole. Table 2 shows the total number of cankers, their locations on the tree (in the branch, at the branch collar on the bole (Fig. 7), or in the bole), and condition (live or dead) for each measurement year. The number of living branch cankers declined during the study until all branch cankers either reached the collar or bole, or died before reaching the bole. Data on 39 cankers were not available for analysis (NA) because the trees or branches on which they occurred had been killed by other cankers before they had time to reach the bole or because the canker had merged with another canker. Three of the five branch cankers that were still alive in 2009, had died by 2010 and the other two had reached the bole. The two that reached the bole were initially 17 and 23 inches from the bole in 2000.

Figure 7. White pine blister rust canker at the branch collar.

Table 2. Total number of cankers, their locations on the tree, and condition over the study period.

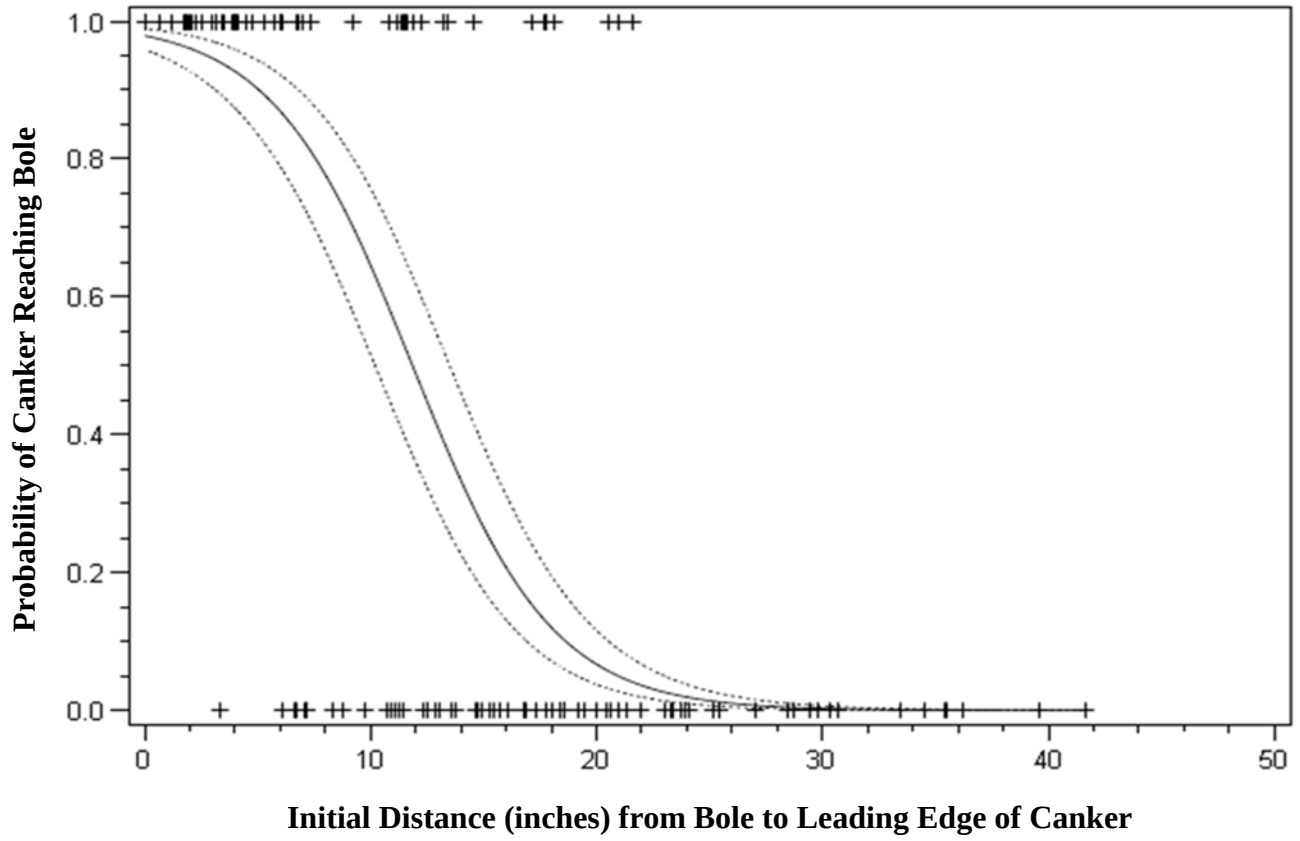
Year	Live Branch	Live Collar	Live Bole	Dead	NA*	Total
2000	113	5	5	0	0	123
2001	164**	3	17	5	2	191
2002	122	20	21	27	1	191
2003	96	24	30	42	0	192
2004	69	24	44	56	0	193
2005	42	21	60	70	0	193
2006	22	23	66	81	1	193
2007	13	14	76	83	7	193
2008	9	6	68	85	25	193
2009	5	7	61	86	34	193
2010	0	9	56	89	39	193
2012	0	-	-	-	-	-

*Some cankers were not available for analysis (NA) because they merged with other cankers, or other cankers killed the bole or branch on which they occurred before they had time to reach the bole. ** In 2001, 68 new branch cankers were added to the study; in 2003–4, 1 new canker was added each year to replace cankers on broken-off branches.

Figure 8 shows the proportion of cankers that reached the bole from various distances out on the branch using data from 2001 through 2009. We included branch collar infections with bole cankers for this analysis since over 97% of cankers that reached the branch collar became bole cankers. Nearly 90% (40 of 45) of the branch cankers that were six inches or less from the bole reached the bole in 3–4 years, but

the proportion reaching the bole declined with distance out. Cankers that started 12 inches from the bole had a 50% probability of reaching the bole within the eight years shown. The probability of reaching the bole within eight years dropped to 10% for cankers at 18.5 inches from the bole. Of the 41 cankers that started farther than 20 inches from the bole, only five caused bole infections and two of those didn't occur until after 2009. None of the 25 cankers that started farther than 24 inches from the bole made it to the bole. In our study, most cankers were 4-5 years old when their margins were first marked with tacks, so the point of origin may have been one to three inches more than our starting distance. The relationship of bole infection to initial distance from the bole is similar to that reported for non-resistant stock. Slipp (1953) reported that only 8 of 321 branch cankers (2.5%) grew more than 24 inches from their point of origin, which substantiated the supposition that only cankers that originated less than two feet from the bole posed a threat to the tree.

Figure 8. Probability of branch cankers reaching the bole or branch collar by initial distance out (2001–2009), with 95% confidence interval[1].



[1] Hash marks at the top of the graph indicate the initial distance out for cankers that reached the bole and those at the bottom indicate the initial distance out of cankers that failed to reach the bole.

Annual monitoring of cankers made it possible to document if and when cankers became inactive or died. We found that most branch cankers die before reaching the bole, and that canker "inactivation" becomes more likely with increasing distance of origin from the bole and with canker age. Our inactivation rate was 62.7% for branch cankers which is similar to the rate reported by Kimmey (1969). He found that 62% of 1,918 cankers examined in 1966 were inactive. However, Hungerford (1977) reported an average inactivation rate of only 37% (varied from 19 to 56%) during seven years of monitoring more than 1,500 cankers. Hungerford also reported an average six percent increase per year in inactivation of cankers

monitored for seven years. During the first six years of this project there was a 7.8% average increase in inactivation per year of branch cankers. Although many cankers became inactive when the branch beyond the canker died and all foliage was lost, others continued to expand toward the bole, and the branch between the bole and the canker remained alive (i.e. cambium was alive but no foliage was present). Most of these situations occurred where the canker was close to the bole, and most succeeded in reaching the bole. For this reason, it is important to remove apparently dead branches (those with no foliage) as well as living ones when pruning.

Canker Expansion Rate

The second objective of the study was to determine the annual rate of expansion of cankers toward the stem. Canker expansion rates for non-resistant trees were reported by Buchanan (1938), Lachmund (1934), and Slipp (1951). Results for the three studies are quite similar: annual canker expansion toward the bole for branches 0.2 inches in diameter was 1.2–1.4 inches, and the rate increased with branch diameter. Cankers on branches 1.0 inches in diameter grew about 2.0–2.4 inches annually.

Table 3 shows the annual canker expansion rates calculated for the period 2000-2009. Although some cankers grew very little while others grew several inches, the annual expansion over the nine intervals averaged 2.06 inches (1.90–2.23 at 95% confidence). The best predictors of canker expansion toward the bole were initial distance out, branch diameter, branch type (primary or not) and year as a fixed effect. Significantly smaller mean expansion rates occurred during the first three monitoring periods than the following two periods. This may be related to weather conditions affecting tree and canker physiology, branch diameters, diminishing sample size, or other unknown factors. The means presented in Table 3 have been adjusted for initial distance from bole, branch diameter, and whether or not the canker occurred on a primary branch.

Table 3. Mean canker expansion toward the bole for live branch cankers in each of the nine re-measurement intervals.

Year	N	Mean (in)	Std. Error	Lower Bound*	Upper Bound*
2000-1	96	1.75	0.081	1.67	1.83
2001-2	119	1.63	0.074	1.55	1.70
2002-3	94	1.79	0.083	1.70	1.87
2003-4	63	2.25	0.102	2.15	2.36
2004-5	40	2.35	0.129	2.22	2.48
2005-6	21	2.25	0.172	2.08	2.42
2006-7	12	2.08	0.228	1.85	2.31
2007-8	9	2.11	0.264	1.85	2.38
2008-9	5	2.38	0.352	2.03	2.73
Total	**459**	**2.06**	**0.165**	**1.90**	**2.23**

*Upper and lower bound for the mean at 95% confidence.

Bole Girdling Rate

Nineteen of the tagged cankers that started in branches eventually grew into the stem and girdled or killed the tree in 10 years. Trees that appeared to be completely encircled by one or more cankers often did not exhibit a reduction in leader growth or thinning and chlorotic crowns until a year or two before they died. We recorded 99% girdle for trees that appeared to be completely girdled but were still alive. Some trees had apparently healthy crowns for as many as nine years after becoming 99% girdled. These observations show that an F2 plantation that looks healthy from a distance may actually be severely infected, and plantations should be closely monitored to evaluate infection and mortality levels.

Figure 9. Margins of two bole cankers merging to girdle a tree.

Since the time cankers first infected the stem and the time of girdling and death was known, the time it took to kill the tree could be calculated. The average time to girdle/encircle the tree was 5.4 years (range 3-8); small trees were girdled more quickly than large ones. The additional time to death after apparent girdling ranged from 1–5 years. Therefore, the total time to encircle and kill the tree averaged 8.8 years (range 6–12). Buchanan (1938) reported the number of years required for girdling "larger" stems was equivalent to the number of inches of stem diameter. Although similar to our 8.8 year average for trees that were 6–8 inches in diameter at death, the results are not directly comparable because our diameters were measured at breast height, whereas Buchanan's were at the canker.

Figure 10. "Abnormal" bole canker on an improved western white pine showing irregular margins and sunken, dark brown bark reaction.

Twenty trees had bole infections at the time of study establishment in 2000 and 2001. Of these, 10 were dead by 2009, but eight others survived for 12 or more years. Three of these eight trees were girdled 95-99%, three others were 88-90% girdled, and the other two were 80% and 65% girdled. By the end of the study a total of twenty-seven trees had bole infections but were still alive. In 2012, these trees ranged from 5-99% girdled. Seventeen of these were more than 80% girdled, and only two were less than 40%.

Most trees had multiple stem cankers by the time the trees began to die, often at the same branch whorl where they merged to girdle the stem (Fig. 9). As a result, the trees likely died sooner than they would have if only one branch canker had entered the bole. But observations indicate that this condition is common in severely infected stands.

A few trees had bole cankers that appeared "abnormal" with irregular margins and areas of sunken, dark brown or necrotic bark, without the distinct yellow margin (Fig. 10). This is frequently referred to as "bark reaction" and is thought to slow

pathogen growth. In fact, the two original bole cankers that had girdled only 80 and 65% of the tree by 2012 were described as abnormal. Bark reaction is desirable in breeding programs because it is thought to be controlled by several genes. Most trees that abnormal cankers also had normal looking cankers. Eckert (2007) examined F2 western white pines with abnormal cankers with some of these same characteristics and found that some cankers girdled at a much slower rate than others.

Canker Sporulation

The total number of sporulating cankers (cankers with aecia) on monitored trees varied from 769 in 2001 to less than 10 from 2007–2010. Although some trees had as many as 70 sporulating cankers in one year, spore production from individual cankers was very sporadic. Most cankers produced characteristic blisters with aeciospores (Fig. 4) for only 1-3 years. Many cankers never sporulated, while others sporulated after two or more consecutive years without sporulation. The average number of years that monitored cankers sporulated was 1.2 years. However, in most cases cankers remained active and continued to expand even in absence of spore production. The reason for poor sporulation of individual cankers is unknown, but may be related to annual washing of cankers, action of secondary insects or fungi, or possible rust resistance. The drop in total numbers of sporulating cankers is probably largely due to the high level of inactivation of cankers due to the stand reaching crown closure and lower branches with most cankers being shaded out.

CONCLUSIONS

Pruning in F2 western white pine plantations to reduce the number of branch cankers as well as the potential for new infections should continue to have positive results. We found F2 canker expansion rates and likelihood of bole infection to be similar to those reported from earlier studies on non-resistant trees. Trees with cankers within six inches of the bole should continue to be considered to be “lethally infected,” those between six and 24 inches as “prunable” cankers, while cankers beyond 24 inches can be considered as “safe” (highly unlikely to reach the bole). In addition, cankers on branches without foliage and those that sporulate intermittently often continue to expand toward the bole, and therefore should continue to be removed in pruning operations. Based on the findings from this study, there is no need to make changes in existing pruning guidelines.

It should be noted that our results come from a severely infected stand and that seedlings from F2 improved seed are a mixture of individuals that can vary greatly in their resistance and response to rust pressure. Our results in no way suggest a level of damage that will occur in all or even most F2 plantations. Many F2 stands have little infection, and in all cases where F2 and non-resistant white pines were compared, infection rates were always lower for F2 stock (Kearns et al. 2012). Infection in our stand, believed to be above 90%, is at or near the upper range of what has been found in young stands (Muller 2002). Yet, high infection has been reported in earlier studies. Fins et al. (2001) reported that one stand (Merry Creek) had 93% infection at 26 years of age, and Kearns et al. (2012) found one stand with 84% infection at 21 years. Many stands with low infection levels may contain moderately to severely infected individual trees, which will likely be killed. Tree girdling and mortality rates in lightly infected stands may be less than what we observed because trees in lightly infected stands may have lower numbers of cankers per infected tree and fewer lethal cankers. Also, there may be fewer merging cankers at the bole, resulting in slower girdling. Nevertheless, branch canker expansion rates and bole infection probabilities, the primary objectives of this study, are likely similar in F2 plantations regardless of amount of infection.

There are still questions that could be answered by additional monitoring. One is the mortality that might be expected in lightly infected trees and plantations. Also important is an understanding of slow canker

growth as a resistance mechanism in F2 stock in operational plantations. In addition, the long-term benefits of pruning F2 stock have not been adequately documented. It seems reasonable to conclude that F2 stock will respond as well or better than non-resistant stock, but this needs to be fully investigated.

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